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THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

Volume 30 June 15, 1946 Number 6

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CHECK LIST REVISION

Freeman Weiss

TRIFOLIUM (LEGUMINOSAE)

TRIFOLIUM AGRARIUM L., HOP CLOVER (1), T. PROCUMBENS L., LOW H.C. (2), and T. DUBIUM Sibth., LEAST H.C., sometimes called "true shamrock" (3). Annuals of Europe, more or less widely naturalized in the U.S., chiefly in the East and South East; (1) occasionally west to Iowa and (3) especially in the Pacific Northwest. At least (2) is of appreciable food value to wildlife.

Cercospora zebrina Pass., leaf spot. N.J., N.Y. (1); Wis. (3)
Colletotrichum destructivum O'Gara, anthracnose. N.J. (2)
Erysiphe polygoni DC., powdery mildew. Conn., Ill., La., N.Y., Pa. (1); Ark., Ga., Md., Miss., N.Car. (2); Ark., Ga., La., Miss. (3)
Péronospora trifoliorum DBy., downy mildew. Oreg. (3)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (2)
Uromyces minor Schroet. (I,III), rust. Calif., Oreg., Wash. (2,3); Okla., Tex., (2), probably also Ala., Ind., Miss. though reported as U. trifolii (Hedw.f.) Lév.

TRIFOLIUM INCARNATUM L., CRIMSON CLOVER (1). European annual grown as winter cover crop and for soil improvement chiefly on the Atlantic and Gulf Coastal Plain and locally in the Central States; also occasionally naturalized. T. ARVENSE L., RABBIT-FOOT C. (2). European annual, naturalized in old fields and waste ground in the Eastern and Central States; furnishes food for wildlife. T. ALEXANDRINUM L., EGYPTIAN C. (3). Annual of the Mediterranean region, cult. as a winter cover crop in Ariz. and southern Calif.

Botrytis sp., gray-mold leaf blight and stem rot. Del., Ky. (1)
Cercospora zebrina Pass., leaf spot. Del., La., Md., Miss., Tenn. (1)
Colletotrichum trifolii Bain & Essary, southern anthracnose. Ky., N.Car. (1)
Cymadothea trifolii (Pers. ex Fr.) Wolf (Polythrincium trifolii G. Kunze), sooty blotch. Me. to Ala. and La., N.Dak. (1)
Ditylenchus dipsaci (Kuehn) Filip., stem nematode. N.Y. (2)
(Dothidella trifolii (Pers. ex Fr.) Bayl.-Elliott & Stansf.): Cymadothea trifolii.
Erysiphe polygoni DC., powdery mildew. Ga., La. (1)
Heterodera marioni (Cornu) Goodey, root knot. Fla., S.Car. (1); Calif. (3)
Kabatiella caulivora (Kirchn.) Karak., northern anthracnose. N.Car., Va. (1)
Phyllosticta sp., leaf spot. Tenn. (1)
Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (1)
(Polythrincium trifolii G. Kunze ex Fr.): Cymadothea trifolii
Pseudoplea trifolii (Rostr.) Petr., leaf spot. Ill., Mo., N.Y. (1)
Pseudomonas syringae v. Hall (P. trifoliorum L.R. Jones, Williamson,

Trifolium cont.

Wolf & McCul.), bacterial leaf spot. Ind., Iowa, Md., N.Car., Va., Wis. (1); Md. (3)

Pyrenopeziza sp., leaf spot. Md. (2)

Ramularia sp., leaf spot. Ga. (1)

Rhizoctonia sp., root rot. N.Car.

Sclerotinia trifoliorum Eriks., Sclerotinia wilt, root rot. N.J. to S.Car. and Tenn., Oreg. (1)

Sclerotium rolfsii Sacc., southern blight, stem rot. La. (1)

Stemphylium sp., leaf spot. Va.

Thielaviopsis basicola (Berk. & Br.) Ferr., black root rot. Wis.

Uromyces trifolii (Hedw. f.) Lév. var. fallens (Desm.) Arth. (0,I,II, III), rust. Del., S.Dak. var. trifolii-repentis (Liro) Arth., Oreg., Wis. This sp., without designation of var., is also reported from Ala., Ind., Minn., Mo. and Pa.

Mosaic -- virus(es). Conn., Ind., N.J., Pa., Va. Viruses infective by artificial inoculation to crimson clover include those of yellow mosaic of beans, common and enation mosaic of peas, pea mottle, pea wilt, pea streak, alfalfa mosaic, and red clover veinal mosaic, also the cucumber mosaic and the potato yellow dwarf viruses. These viruses have not usually been differentiated in natural infections, but crimson clover has been shown to be a natural host of yellow bean mosaic in Oreg.

TRIFOLIUM PRATENSE L., RED CLOVER (1), European perennial widely cult. for hay and forage, and naturalized in agricultural areas throughout the U.S. Several vars. as PERENNE and SEROTINUM, sometimes called MAMMOTH CLOVER, are distinguished. T. MEDIUM L., ZIGZAG C. (2), of similar origin and habit, is only occasionally cult. here.

Alternaria tenuis Nees ex Wallr., seed mold. Cosmopolitan. Secondary leaf spot, Va. (1), Vt. (2)

Ascochyta spp. (doubtfully reported as A. pisi Lib. and A. imperfecta Pk.; perhaps A. trifolii Bond & Trusova = Stagonospora recedens, q.v.), leaf spot. Ill., Wash. (1). See also Phoma.

Botrytis cinerea Pers. ex Fr., gray-mold leaf blight, stem rot. N.Y., Pa. (1)

Cercospora zebrina Pass., leaf spot. Me. to Md., Mo., and Minn. (1); N.Y., Vt. (2)

Colletotrichum trifolii Bain & Essary, southern anthracnose. General from N.J. to Ala., Tex. and Ill.; occasionally reported north and west as N.Y., Mich., Wis., Wash. (1). C. destructivum O'Gara, producing similar symptoms, has been reported in Ky., Minn., and Utah; C. graminicolum (Ces.) G.W.Wils. in Ky. and Ohio; all on (1).

Cuscuta spp. (especially C. arvensis Beyrich and C. epithymum Murr.), dodder. General in the Eastern and Central States and southward,

Trifolium cont.

rare in the West.

Cylindrocladium scoparium Morgan, stem rot (? secondary). Tenn. (1)

Cymadothea trifolii (Pers. ex Fr.) Wolf, sooty blotch. Me. to N.Car., Tex. and Wash. (1)

Didymella trifolii (Fckl.) Sacc., on stems. Miss. (1)

Ditylenchus dipsaci (Kuehn) Filip., stem nematode. Calif., Ida., Oreg., Utah, Wash. (1)

Erysiphe polygoni DC., powdery mildew. General (1); Ky., N.Y., Vt. (2). Commonly found in conidial stage only; perithecial stage reported in Ida., Mont., N.Y., Oreg., Wash., W.Va. Was rare in the U.S. prior to about 1920.

Fusarium spp., root rot (? secondary). Ind., Ky., Minn., N.Y., Ohio (1). The following spp. have been identified: ? F. graminearum Schwabe, Ohio; F. moniliforme Sheldon, F. oxysporum Schlecht., and F. solani (Mart.) App. & Wr. in Ky.

(Gloeosporium trifolii Pk.): Sporonema t.

Heterodera marioni (Cornu) Goodey, root knot. La., S.Car., Utah, W.Va. (1); also reported on (2)

Hoplolaimus coronatus Cobb, root nematode. Md. (1)

Kabatiella caulivora (Kirchn.) Karak., northern anthracnose. Me. to Va., Mo., and Oreg. (1)

Macrophomina phaseoli (Maub.) Ashby, charcoal rot. Ky. (1)

Macrosporium. See Pleospora and Stemphylium

Microsphaera alni DC. ex Wint., powdery mildew. N.Y. (1)

Peronospora trifoliorum DBy., downy mildew. N.Y., Wyo. (1)

Phoma spp., leaf and stem spot, black stem. P. medicaginis Malb. & Roum. (= Ascochyta imperfecta Pk. ?) reported from Ida., and P. trifolii E. M. Johns. & Valleau from Ky. and Wis., but the precise position of these in the Asochyta-Phoma-Stagonospora complex on Leguminosae is not yet determined. Black stem of undetermined cause is also reported from N.Y.

Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (1)

Pleospora herbarum (Pers. ex Fr.) Rabh. (Stemphylium botryosum Wallr.), seed mold, secondary leaf spot. Cosmopolitan.

Pratylenchus pratensis (DeMan) Filip., root nematode.

Pseudomonas syringae v. Hall, bacterial leaf spot. N.Y. to N.Car., Iowa, and Minn. (1); N.Car., Va., Wis. (2)

Pseudopeziza trifolii (Biv.-Bern. ex Fr.) Fckl., leaf spot. Me. to Md., Nebr., and the Pacific Northwest; also Calif. and Alaska (1); N.Y. (2)

Pseudoplea trifolii (Rostr.) Petr., leaf spot. Ill., Kans., Mo., N.Y. (1)

Rhizoctonia crocorum DC. ex Fr. (= Helicobasidium purpureum (Tul.) Pat.), violet root rot. Oreg. (1)

Sclerotinia trifoliorum Eriks., Sclerotinia wilt, root rot. N.Y. to Miss., La., and Mich.; Oreg., Wash. (1); Oreg. (2).

(Sclerotium bataticola Taub.): Macrophomina phaseoli.

Septoria trifolii Ell., on dead leaves. N.J. (1)

Trifolium cont.

Sporonema trifolii (Pk.) Greene, leaf spot. Mich., N.Y., Ohio, Wis. (1)

Stagonospora recedens (Massal.) F.R.Jones & Weimer, leaf spot. Calif., Iowa, Wis. (1). S.meliloti (Lasch) Petr., and its synonym S. dearnessii Sacc., as reported on this host in N.Y. and Wis., probably belong here; perhaps also some of the fungi reported as Ascochyta spp.

Stemphylium sarciniforme (sarcinaeforme) (Cav.) Wiltshire, leaf spot. Me. to N.Car., La., Kans. and N.Dak. (1); Vt. (2)

Thielaviopsis basicola (Berk. & Br.) Ferr., black root rot. Conn., N.Y., N.Car. (1)

Uromyces trifolii (Hedw. f.) Lév. var. fallens (Desm.) Arth. General (1); Ind., Md., Nebr., N.Y., S.Dak. (2)

Black stem. See Phoma.

Girdle -- stem canker of undetermined cause. N.Y., ? Ida. (1)

Mosaic -- virus (probably mostly Marmor leguminosarum Holmes, Pisum virus 2 K.M.Sm.; also the pea mottle and pea wilt viruses, singly or together). Widespread but infrequent. The following viruses also are infective to red clover by artificial inoculation but have not been differentiated in this host: alfalfa mosaic (including the potato calico strain), yellow mosaic of bean, and pea streak.

Ring spot -- virus (? Marmor annularium McK., Nicotiana virus 12 K.M.Sm. -- yellow strains). Ky. (1)

Veinal mosaic -- virus (Marmor trifolii Holmes). Ill., N.J. (1)

White spot -- physiological, perhaps calcium deficiency. Ill., N.J. (1)

Yellow dwarf -- virus (Marmor vastans Holmes var. vulgare Black = Aureogenus vastans Black, Solanum virus 16 K.M.Sm.). N.Y. (1) Also susceptible to the related viruses Aureogenus v. var. agalliae Black and A. magnivena Black.

Yellows -- in part physiological, probably potassium deficiency. Ark., Conn., Ill., N.J. (1). In part due to leafhopper injury. N.J., Va., Wis. (1)

TRIFOLIUM REPENS L., WHITE CLOVER (1). Perennial of Europe, cult. in pastures and lawns, a prime honey plant, naturalized throughout the U.S. except the arid plains. The Ladino variety, a robust type, is cult. in the northeast, the southern Piedmont, and the Pacific Northwest. T. HYBRIDUM L., ALSIKE CLOVER (2). Similar, but larger and more upright, is cult. for forage and mixed with grasses, for hay; widely naturalized but not common. T. CAROLINIANUM Michx., CAROLINA C. (3). Perennial of the Southeastern and the Southern States west to Tex.; furnishes food for wildlife.

Ascochyta sp., leaf spot. Me., Mass., Vt. (1); Ill. (2). See also Stagonospora.

Trifolium cont.

Blennoria trifolii Bonar, on leaves with Curvularia, probably secondary. Md. (1)

Botrytis sp., gray-mold blight. Ala. (1)

Cercospora zebrina Pass., leaf spot. N.Y. to Ala., La., and Wis. (1 & 2); Utah (1)

Colletotrichum spp., anthracnose. Specific reports as follows: C. destructivum O'Gara, Utah (2); C. graminicolum (Ces.) G.W.Wils., Ky. (1); C. trifolii Bain & Essary, Va. to Ga., Tex., and Ill.; ? Me., N.Y., Wash. (1)

Curvularia trifolii (Kauff.) Boed., leaf spot. Md. (1)

Cymadothea trifolii (Pers. ex Fr.) Wolf, sooty blotch. General (1 & 2). Reputed to be the cause of "trifoliosis", or mouth ulcers, in animals grazing on infected plants.

Ditylenchus dipsaci (Kuehn) Filip., stem nematode. Nev., N.Y. (1)

Erysiphe polygoni DC., powdery mildew. Widespread but infrequent -- Vt. to Fla. and Mo. (1); Vt. to S.Car., Ill., and Minn. (2); Pacific Northwest (1 & 2).

Fusarium sp., root rot. Ala. (2)

F. moniliforme Sheldon, F. oxysporum Schlecht., and F. solani (Mart.) App. & Wr., Ky. (1)

Heterodera marioni (Cornu) Goodey, root knot. Ala., Calif., W.Va. (1); occasionally on (2)

H. schachtii A. Schm., root nematode. Utah. (1)

Olpidium trifolii (Pass.) Schroet., leaf curl. La. (1)

Peronospora trifoliorum DBy., downy mildew. Nebr. (1)

Phymatotrichum omnivorum (Shear) Dug., root rot. Tex. (1)

Physarum cinereum Batsch ex Pers., slime mold. Ind., Md., Pa.

Pseudomonas syringae van Hall, bacterial spot. N.Y. to N.Car., Nebr., and Minn. (1 and 2).

Pseudopeziza trifolii (Biv.-Bern.) Fckl., leaf spot. Calif., Mass., Miss., Mont., N.Y., Oreg., Vt. (1); N.Y., W.Va. (2)

Pseudoplea trifolii (Rostr.) Petr., pepper spot, leaf scorch. Northeastern States to La. and Wis. (1); Ill., Mo., N.Y. (2); Ala. (3)

Rhizoctonia solani Kuehn, brown patch, root rot. N.Y., Ohio, Wis.; probably general in humid regions (1); Minn., Ohio (2)

Sclerotinia trifoliorum Eriks., stem canker, root rot. Ala., Kans., Ky., Miss., N.Y., Oreg. (1); Mich., N.Car., W.Va. (2)

Sclerotium rolfsii Sacc., southern blight. Ala.

Stagonospora meliloti (Lasch) Petr. (S. dearnesii Sacc.), leaf spot. Ala., Ida., La., N.J., N.Y., Oreg., W.Va., Wis. (1); Mont., N.Y., Wis. (2). Conidial stage of Leptosphaeria pratensis Sacc. & Briard, not yet found on this host. Reports of leaf spot caused by Aschochyta sp. may belong here.

Synchytrium sp., leaf gall. ? Tex. (3)

Stemphylium sarciniforme (Cav.) Wiltshire, leaf spot. Ind., N.Y., Vt. (2)

Thielaviopsis basicola (Berk. & Br.) Ferr., black root rot. Conn.,

Trifolium cont.
Tex. (1 & 2).
Uromyces elegans (Berk.) Lagh. (I,III), rust. S.Car. to Fla. and Tex. (3)
U. nerviphilus (Grognot) Hotson (0,I,III), rust. Colo., Mich., Mont., Nev., N.Y., Wash., Wis. (1)
U. trifolii (Hedw. f.) Lév. var. trifolii-repentis (Liro) Arth. (0, I,II,III), rust. Me. to N.Car., Mo., and Tex.; Calif. and Pacific Northwest. (1)
U. trifolii var. hybridi (W.H.Davis) Arth. (0,I rare, II,III), rust. On (2) with range like (1) above.
Urophlyctis trifolii (Pass.) Magn., leaf gall. La., S.Car.

Girdle (stem canker) -- cause unknown. Mo.
Mosaic -- virus(es). Widely distributed but infrequent. Formerly attributed to a specific virus, Trifolium virus 1 K.M.Sm.; now shown to be due sometimes and perhaps generally to the joint action of 2 viruses, Marmor efficiens F.Johns. and M.repens F.Johns. Susceptible by artificial inoculation also to the following viruses: alfalfa mosaic, yellow bean mosaic, pea streak, red clover veinal mosaic, potato yellow dwarf, and ? tobacco ringspot, which have not been differentiated in these hosts. A mosaic of alsike clover has been attributed to a specific virus similar to yellow mosaic of beans, or perhaps to the 2 viruses found in white clover.
Pseudo-mosaic -- genetic abnormality causing variegation. Pa. (1)
White spot -- physiological, perhaps calcium deficiency. Va. (2)

TRIFOLIUM spp. Miscellaneous native spp., annual or perennial, occurring chiefly in the mountain areas of the Western States, in alpine meadows or in open or wooded uplands, affording limited grazing for livestock and wildlife; as T. KINDLERI Greene (1), T. PARRYI A. Gray (2), T. RYDBERGII Greene (3), T. VARIEGATUM Nutt. (4), T. WORMSKJOLDII Lehm. (5), and others (spp.).

Cymadothea trifolii (Pers. ex Fr.) Wolf, sooty blotch. Calif. (5, spp.), Nev. (1), N.Mex. (1,3), N.Dak. (spp.), Oreg. (5).
Erysiphe polygoni DC., powdery mildew. Colo. (5), Ida. (spp.), Mont. (4), Oreg. (5, spp.), Utah (3), Wash. (spp.)
Leptosphaeria monticola Ell. & Ev., on leaves and stems. Colo. On T. kingii S. Wats.
Pleospora herbarum (Pers. ex Fr.) Rab., on leaves. N.Mex. (spp.)
Thecaphora deformans Dur. & Mont., seed smut. Calif. On T. tridentatum Lindl.
Uromyces minor Schroet. (0,I,III), rust. Calif. (4,5,spp.), Colo. (1,2,3,spp.), Mont. (3), Nev. (1,4), Oreg. (spp.), Utah (2,spp.), Wash. (4,spp.), Wyo. (2,spp.)

TRIGONELLA (LEGUMINOSAE)

TRIGONELLA FOENUM-GRAECUM L., fenugreek. Annual of Mediterranean region, used for forage and stock food but rarely cult. in the U.S.

Entyloma sp., leaf smut. Calif.
Heterodera marioni (Cornu) Goodey, root knot. Calif.

DIVISION OF MYCOLOGY AND DISEASE SURVEY

THE DISTRIBUTION OF PARASITIC NEMATODES ON ECONOMIC PLANTS IN IDAHO

Earle C. Blodgett[1]

Plant diseases caused by nematodes have been the subject of repeated studies and attempts at control in certain areas of the United States, particularly in the South. According to Steiner (10) the first publications in the United States on plant parasitic nematodes appeared in 1889, and since that time a large bibliography has been built up. The subject still merits far more attention than it receives. Surveys conducted by the Emergency Plant Disease Prevention Project in 1943, 1944 and 1945, in cooperation with the Idaho Agricultural Experiment Station have shown definitely that more attention should be given to nematode diseases in this State. One of the first requisites to a proper approach to this problem is information on the historical background, the distribution so far as known and the apparent trend in importance of these diseases. For convenience, the different diseases will be treated separately and in the order of their discovery in Idaho.

The Stem Nematode, Ditylenchus dipsaci

Alfalfa and clover have no doubt been grown in Idaho from the time of the early settlers, around 1870, and at the present time alfalfa is one of the most important crops grown in the State, the acreage production 1935-1939 being 1,894,000 tons[2]. Seed production of both crops is an important industry.

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[1] Formerly Plant Pathologist, Emergency Plant Disease Prevention Project, U.S.Department of Agriculture, Moscow, Idaho. Part of the information presented was secured while the writer was located at Aberdeen, Idaho and employed by the Idaho Plant Pest Control and Research Commission. The writer wishes to express his sincere appreciation to Gerald Thorne, John L. Toevs, E. W. Whitman and others who in many ways assisted in securing the information and read and criticized the manuscript.

[2] These data were furnished through the courtesy of the office of Richard C. Ross, Agricultural Statistician, U.S.Department of Agriculture, Boise, Idaho.

According to Smith (8) the first report of the stem nematode in North America was made in 1907 in rye from Kansas. In Idaho it was found first in southern Idaho in 1919 on clover, but farmers stated that it had been present for several years previous (1913-1914 at least). At the time of Smith's work (8,9) the disease caused by it was regarded as not only very destructive but as rapidly spreading and increasing in severity, and was commonly known as "winter kill" of clover.

It was mentioned as being "generally distributed in most of the large clover growing sections of the State."

It should be pointed out that it is generally believed that the stem nematode attacking clover is different biologically but not morphologically, from the one attacking alfalfa; onion, strawberry, etc. Although the species (dipsaci) has a wide host range, including also the following crops, no records are known of any form attacking grain crops, strawberries, or onions in Idaho. In experimental tests Smith (8) noted injury to bean seedlings. Godfrey (5) stated that the alfalfa stem nematode (Tylenchus dipsaci) was observed in America in 1922 and 1923 in isolated localities. He reported that "in a small experimental way red, white and alsike clovers, buckwheat, rye, English pea, turnip and even potatoes, have been attacked and definitely injured by the alfalfa eelworm."

Although no new records of infestations by the stem nematode are indicated, the following list shows the locations and hosts involved as found during the survey:

1. Ladak alfalfa -- Caldwell -- May 1945
2. Common alfalfa -- (Field 1) -- Caldwell -- May 1945
3. Common alfalfa -- (Field 2) -- Caldwell -- May 1945
   These three collections were made about 10 miles east of Marsing on the north side of the Snake River.
4. Common alfalfa -- Mountain Home -- May 1945
   Red clover plants in fields 3 and 4 were not infected.
5. Alfalfa
   In 1937, C. W. McDuffie, U.S.Department of Agriculture, Bureau of Entomology and Plant Quarantine, collected 8 lots from near Parma, Roswell, Greenleaf, and Caldwell, 6 of them being infected with Ditylenchus dipsaci[3].

Of considerable interest in respect to nematodes are specimens of an onion disease in a commercial crop at Mesa, Idaho, collected May 1945 by

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[3] These determinations and the confirmation of diagnosis in specimens 1 to 4 were made by Gerald Thorne, U.S.Department of Agriculture, Salt Lake City, Utah.

Glenn KenKnight of the Idaho Agricultural Experiment Station. Although a Fusarium bulb rot was present and finally was regarded as the primary cause, an extremely high population of the nematode Panagrolaimus subelongatus was consistently present.[4]

It would appear from the evidence secured during the survey that the stem nematode, while not causing serious damage, is still an important disease factor and it is believed by the writer that more careful surveys and closer observations will reveal more injury than is now thought to exist.

The Sugarbeet Nematode, Heterodera schachtii

Sugar beet production in Idaho dates from about 1903 when the first factory was built at Sugar City in the Upper Snake River Valley. It is likely that the industry started somewhat earlier in southeastern Idaho near Preston and Franklin in Cache Valley. Factories were built at Burley in 1912, Twin Falls in 1916, and Paul in 1917. With the development of curly top-resistant selections, the beet area was enlarged and the Nampa factory was built in 1942. A factory built at Nyssa, Oregon in 1938 also depends largely on beets from adjacent areas in Idaho. Plantings in the State of Idaho average about 58,000 acres per year but only 43,000 were planted in 1944 and 53,000 in 1945. Of special significance is the value of sugar beets in crop rotations and in by-products for stock feeding.

With the exception of curly top, nematode injury is regarded as the principal disease involved in sugar beet culture in Idaho. In personal correspondence with Gerald Thorne, he states that, "Sugar beet nematodes were present in many fields near Franklin and Preston, Idaho when I made the first surveys in 1918-1919, indicating that the pest had been there 15 or 20 years. Much the same situation existed at Sugar City, Idaho when I first visited that section in 1928; many large fields were almost completely infested, indicating that nemas had been there for a long time."

According to the best information available sugar beet nematode distribution in Idaho is as follows:

1. There are reported to be 50 to 75 field infestations near the Franklin factory and near Preston and Whitney; one severe case was seen by the writer in 1944.

2. The most generally infested fields are near the factories at Blackfoot, Idaho Falls, Shelley, and Sugar City, with many others

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[4] These determinations were made by Gerald Thorne, U.S.Department of Agriculture, Salt Lake City, Utah.

in the surrounding areas. Field men refuse to contract beets for the second crop year on land known to be infested and suitable crop rotation is an absolute necessity in many cases. In infested areas, crop rotation is set up preferably on a 5-year and at least on a 3-year rotation with strict observance of no consecutive beet crops. In the Sugar City area the rotations are 7 and 5 years. One infested field has been reported near Lewisville, apparently the northernmost location.

3. Field men at Burley reported that they knew of 2 affected plantings in 1943, that there were likely about 10, and that in the Burley, Paul and Rupert area approximately 30 fields were known to be infested.

4. In the large area from Murtaugh to Buhl on the south side of the Snake River and from Hazelton to Gooding on the north side, only one infestation is reported. It is near the factory at Twin Falls and was observed by the writer and Albert Murphy in September 1943.

5. In the rapidly expanding and extensive farming area of the Boise and Snake River Valleys from Boise to Weiser and from Marsing to Ontario, Oregon, no infestations are known at present.

It is evident that the sugar beet nematode is a formidable pest. The Idaho manager of the Utah-Idaho Sugar Company, Mr. Paul Scalley, in connection with the survey has informed the writer that the loss in 1943 from the sugar beet nematode in eastern Idaho and Garland district of Utah has been estimated at 10,247 tons.

The most significant factor is the localization of the pest in eastern Idaho and the grave menace it poses for the sugar beet areas farther west. Fortunately beets are not being moved from infested to clean factory districts and every effort should be made to see that this is not done in the future.

The most common method of distribution, once it becomes established in a locality, is by means of dump dirt from the loading stations. This is especially dangerous where trucks haul for several growers at the same time carrying the dump dirt from farm to farm. Not only nematodes but noxious weeds, fungus diseases, and other soil borne pests and diseases may be transported in this manner.

Special methods and means of handling this dump dirt are being devised and it is believed that eventually the actual savings will be greater than the cost. In addition there will be the advantage of protecting clean fields and areas from infestation.

The Rootknot Nematode, Heterodera marioni

According to Bennett (1) the first record of acreage and production of potatoes in Idaho was made in 1882. At that time 2,000 acres were grown with a production of 250,000 bushels. In 1914 W.L.Carlyle, Director of the Idaho Agricultural Experiment Station (11), stated that "The potato has been one of the leading crops grown in Idaho for several years and should continue one of the most profitable crops in the future." This prediction has been realized with most of the increase occurring after about 1920. In 1943 the production was 43,700,000 bushels according to Richard C. Ross, Agricultural Statistician, Boise, Idaho. Mr. C. E. Temple, Plant Pathologist for the Idaho Agricultural Experiment Station, writing on "Potato Diseases and Insect Pests" (11), called attention to the danger of root knot nematodes and the "recent, 1913" reported outbreaks in California and Arizona. He warned that infected seed will spread the disease and requested that suspected occurrence in the State be reported to the Experiment Station "for the pest must not be allowed to spread."

In 1922, however, root knot-infested tubers were collected in lots grown in Canyon County near Wilder. At that time this area was an important early potato section. Although at the moment considerable anxiety was expressed by regulatory officials no attempts toward control were made. Potato growing in this area fell off (but for other reasons) and no further reports on root knot were made in the State until about 1935 when severe infestations were found in eastern Idaho near Fort Hall and Idaho Falls. Information was made available to growers through mimeo-leaflets (7) published by the Idaho Agricultural Experiment Station.

There is no good explanation why root knot has not flourished in Canyon County since row cropping is widely practiced. In 1943 one lettuce field near Nampa was found by the writer to be heavily damaged by root knot and in 1944 a collection was made on carrots and turnips in a garden at Hammett (Elmore County). Gerald Thorne (personal correspondence, 1945) stated that a field of sugar beets near Nampa was very severely damaged by root knot.

It is pertinent to note too that in 1945 a large shipment into Idaho of carrot roots for seed production was made by a leading seed company located in Idaho. The roots came from California and since they were reported by Idaho State officials to be severely infected by root knot they were ordered destroyed. Definite records of root knot infestations in Idaho are as follows:

1. Potatoes, Wilder, Canyon County, 1922 (according to personal communication and reports of the State Department of Agriculture) W. H. Wicks (13)

2. Potatoes, near Chubbuck and Tyhee, Bannock County. 1939-1945.

There are known infestations on 6 farms and one suspected within about 3 miles of Chubbuck and Tyhee. Severity ranges from moderate to very severe. In 2 cases almost complete loss of crops occurred. In addition to the 6 known cases, the Inspection Service claims that there are several slight infestations but exact locations are not known.

3. Potatoes, Fort Hall, Bannock County, 1935-1945.
There are two farms and probably one other that are heavily infested.

4. Potatoes, McDonaldville District, Bingham County, 1945.

5. Potatoes, Lavacide District, Bingham County, 1943-1945.
There are 5 known infested farms.

6. Potatoes, Idaho Falls District, Bonneville County.
There are 7 known infested farms within 5 miles southwest of Idaho Falls. According to the best information available the first infestations were found about 1935 and were reported to be the result of growing potatoes from seed brought in from California. One farm about 6 miles northeast of Idaho Falls is the farthest north location definitely known although a farmer reported root knot on potatoes on a farm about 3 miles southwest of Roberts.

7. Potatoes, Liberty, Pingree District, Bingham County. 1940-45.
Two infested farms are known. A farmer stated that in one field, infestation was traced to the area where manure was scattered after feeding infected culls.

8. Potatoes, Aberdeen area, Bingham County. 1943-1945.
There are 5 known infested farms all with little more than a trace. They are located east, northeast, northwest, and west of Aberdeen and one south near the Power County line.

9. Lettuce, Nampa area, Canyon County, 1943.

10. Carrots, turnips, Hammett area, Elmore County, 1944.

11. Sugar beets, Nampa area, Canyon County, 1945.
(personal communication, Gerald Thorne)

12. Sugar beets, Preston area, Franklin County, 1945.
(personal communication, Gerald Thorne)

Importation of great quantities of ornamental plants, tubers, root stocks, tomato and cabbage plants, etc., have undoubtedly accounted for the presence of this pest in Idaho. Although the root knot nematode is regarded as more at home in southern climates it has demonstrated its

ability to become an important disease-producing organism in this State. At least one case is known where its presence in a field caused a prospective buyer to refuse to purchase the farm. Cases are known where the infestation has actually destroyed the potato crop, and a farmer has stated that he was able to determine the presence of severe damage to the crop by the appearance of the potato vines. Apparently the pest is already well established and Survey records indicate that its distribution and severity are on the increase in some of the best commercial potato-producing areas in the State. Since seed stocks from this general area are widely distributed, it represents a serious source of distribution to other localities.

The Potato Rot Nematode, Ditylenchus destructor

This disease was first observed in Idaho in a field near Aberdeen on October 26, 1943. The first reports following the discovery indicated that the disease was caused by Ditylenchus dipsaci, the stem nematode common on alfalfa and clover, but Thorne (12) showed that the nematode attacking potatoes was a different species, which he named D. destructor.

The writer (2,3) has called attention to special surveys on this pest. As known at present, distribution in the United States is apparently confined to several fields near Aberdeen.[5] The present importance of this disease has resulted in very strict control measures and in plans for intensive research on methods of eradication.

The present status of our information in Idaho indicates that this nematode, while capable of severe damage in certain cases, is apparently near its limits of survival and since the actual loss at present is minor in comparison with that from most other common diseases in the area, its importance is due to its restricted distribution. It is believed that environmental and climatic factors, crop rotation, natural enemies, limited host range, etc., all combine to reduce effectiveness of spread and rapid build-up of the pest. Known infestations near Aberdeen, Idaho involve about 20 fields on 13 farms in 8 sections of land. Most of the infested farms show traces (1 to 3 tubers found) or very slight damage. It is believed from evidence secured during the two seasons since its discovery that the disease can be effectively controlled and possibly eradicated.

General Discussion

Four diseases of economic plants caused by nematodes are discussed in this paper. While they are all potentially grave problems, it is believed that the root knot nematode can become the most serious menace to the

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[5] Recently (December, 1945) its presence in Prince Edward Island, Canada, has been confirmed from specimens submitted by Dr. A. D. Baker, Department of Agriculture, Ottawa, Ontario, Canada. (Gerald Thorne, personal communication).

crops of Idaho. Just how accurate this statement is depends in large measure on the results of future studies on host relationship and parasitic strains. As shown by Christie and Albin (4) there are different strains of the root knot nematode and only by carefully conducted tests can this information serve as a basis for control. The present status of distribution and the ease of spread through potato seed and beet dump soil indicate the importance of timely research and strict observance of adequate control measures. The present and potential distribution of the sugar beet nematode in Idaho (and adjacent Oregon) should emphasize that almost any measures to prevent its spread are economical and desirable. Particular attention is needed in regard to adequate disposal of soil from beet dumps.

Early dying of potato vines in certain areas in fields, in certain fieldsin a locality, and in certain localities has been a problem of increasing importance. It has a rather wide yearly variation and it has been attributed to the work of insects such as leaf hoppers and to soil-borne fungi such as Fusarium (6). The Idaho Agricultural Experiment Station Entomologists have shown quite conclusively that leaf hoppers are not involved in early dying of potato vines. Evidence indicates too that factors causing early dying are not seed-borne.

In September, 1945 the writer became interested in the theory that certain nematodes such as Scribner's meadow nematode or root lesion nematode (Pratylenchus) may be involved. The theory gained support with the recent publication by Steiner (10) of a paper discussing these worms as a cause of root destruction. Of course only circumstantial evidence is available but it has been demonstrated (according to growers) that in fields where carbon bisulfide is used as a weed killer, potato vines stay green longer. Of course this action might result when soil fungi are involved. Both field tests and laboratory soil tests have been started in connection with this theory.

The importance of known plant parasitic nematodes in the State and the probable existence of others seem to justify the establishment of a well supported project to deal with these problems collectively.

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EMERGENCY PLANT DISEASE PREVENTION PROJECT AND IDAHO PLANT PEST CONTROL AND RESEARCH COMMISSION

THE SPREAD AND DEVELOPMENT OF CUCURBIT DOWNY MILDEW IN ATLANTIC COASTAL STATES IN 1945

C. J. Nusbaum

Continuing the cucurbit downy mildew [Pseudoperonospora cubensis] reporting program in 1945, nine cooperators in States from Florida to Massachusetts again assisted the writer in following the course of this disease in Atlantic Coastal States. This was done in an attempt to disseminate timely information on the progressively northward spread of the disease with the end of anticipating the time of its appearance and possible severity in cucurbit producing areas. An analysis of the results for 1943[1] and 1944[2] have been reported previously. A summary of the 1945 results is listed below:

Florida. Downy mildew was observed in and around Manatee County (west-central Florida) throughout the winter months but was less destructive

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[1] Nusbaum, C. J. The seasonal spread and development of cucurbit downy mildew in Atlantic Coastal States. Plant Dis. Reptr. 28 (3): 82-85. 1943.

[2] _____. The seasonal spread and development of cucurbit downy mildew in Atlantic Coastal States. Plant Dis. Reptr. 29 (5): 141-143. 1944.

than usual because of prolonged dry weather. However, an abundance of inoculum was present during April and May.--A. L. Harrison.

The first occurrence of downy mildew in the Gainesville section (north Florida) was observed about April 20th on early cucumbers. By May 1st the disease was widely scattered but still at a relatively low level because of unfavorable weather. By May 15th infection was general in all cucumber plantings.--George F. Weber.

South Carolina. In the Charleston area primary lesions were first found on cucumber on May 9th (about three weeks earlier than usual) following a wet period which undoubtedly favored the early establishment of the disease. Secondary infection was widespread by May 17 and there was as much as 50 percent defoliation in some cucumber fields by May 29th when the harvest was at its peak. Many growers reported serious damage. --C. N. Clayton.

The first infection in the vicinity of Blackville, South Carolina (100 miles north of Charleston) was observed on cucumber on May 25th, about three weeks earlier than in 1944. However, the disease was checked by unfavorable weather until mid-June thus permitting the cucumber crop to escape serious damage. The latter part of June was favorable for downy mildew and cantaloups suffered considerably. Many fields showed almost complete defoliation by July 1st and the harvest season was cut short. In most cases, however, the growers realized good returns because of the heavy demand and high prices. Sporulation continued during July in abandoned fields.--C. J. Nusbaum.

North Carolina. Downy mildew was first observed on both cucumbers and cantaloups in Scotland County (southern coastal plain area) on June 8th about three weeks earlier than usual. At that time infection was light and of recent origin. The disease probably became established about June 1st with the advent of favorable weather.

The disease developed somewhat irregularly, being light in some localities and destructive in others. In the Wilmington section (southern coastal area) cucumbers were heavily infected and defoliated by June 20th. At New Bern, about 100 miles north of Wilmington, the disease appeared much later, primary lesions being observed on cucumbers on June 30th. Following the early establishment of downy mildew in Scotland County, hot dry weather held the disease in check until late June but the return of favorable weather brought about renewed spread and, by July 10th, some cantaloup fields showed as much as 40 percent defoliation. In general, damage was slight in early fields and moderate to heavy in late plantings. In the Ridgeway section (northern, upper coastal plains area) primary lesions were found on cantaloup on July 11th about ten days before harvest began but subsequent development of the disease in this area was not ascertained.--D. E. Ellis.

Delaware. Downy mildew was not observed during the State-wide surveys of July 5th, 19th and 23rd. When another survey was made on July 26th and 27th, however, widespread infection of both cucumber and cantaloup was found. Weather conditions favored the disease and heavy sporulation was noted. Although downy mildew appeared at about the usual time, cucumbers escaped and the extent of damage to cantaloups was not reported. --J. W. Heuberger.

Pennsylvania. Widespread infection was noted in the vicinity of Philadelphia on August 8th. Sporulation was abundant. It was judged that the disease became established during late July.--Richard P. Porter.

Massachusetts. Downy mildew became established about three weeks earlier than usual. On July 26th a single spot (sporulating heavily) was found in a field of cucumbers in Franklin County (Connecticut Valley area). On August 1st downy mildew was observed in one cucumber field in Bristol County, the infection probably having appeared about ten days previously. On August 3 the disease was found in four out of six commercial plantings of cucumbers in Plymouth County and, in these cases, the infection appeared to be about two or three weeks old.

By mid-August infection was quite widespread in cucumber fields in the Connecticut Valley area. Most of the infected fields showed a few foci of primary infection, in which the vines were declining rapidly, and scattered secondary infection of more or less recent origin. On August 16th several cucumber fields in Essex County were examined but mildew was not found. Muskmelon plantings were also mildew-free.--O. C. Boyd.

Summary. The 1945 season was in general characterized by early establishment of cucurbit downy mildew in most commercial producing areas of the Atlantic Coastal States. Where losses were reported following early establishment, delayed harvest and local weather conditions favorable to the disease were important contributory factors. On the other hand the occurrence of conditions unfavorable for downy mildew undoubtedly reduced losses in some areas. The hot, dry spells which soon followed the appearance of the disease in the Blackville section of South Carolina and in certain localities in North Carolina seem largely responsible for checking the early spread of the disease and the subsequent destruction of extensive commercial plantings of cantaloups.

It is interesting to note that downy mildew appeared at Charleston, South Carolina early in May and at Blackville, 100 miles north of Charleston, about three weeks later in spite of the fact that plantings in both localities were generally comparable in their stages of growth and that early May weather conditions in both localities seemed equally favorable for the establishment of the disease. It appears possible that the infection at Charleston arose from spores originating at some point to the south, probably northern Florida, and that the establishment of the

disease at Blackville awaited the production of inoculum in the Charleston area. This case represents an isolated example in support of the view that downy mildew migrates northward by means of wind-borne spores. In the coastal region of North Carolina, where infection at Wilmington appeared at least three weeks earlier than at New Bern, the circumstances might have been similar to those in South Carolina.

It is hoped that collaborators will continue to develop the cucurbit downy mildew reporting service with the view of building a fund of information that will further test the validity of the hypothesis of migratory spread and afford a more reliable appraisal of its true worth in relation to mildew control.

EDISTO EXPERIMENT STATION
BRANCH OF THE SOUTH CAROLINA EXPERIMENT STATION
BLACKVILLE, SOUTH CAROLINA

PLANT DISEASES IN PALM BEACH, BROWARD AND ST. LUCIE COUNTIES, FLORIDA DURING APRIL

G. R. Townsend, R. C. Cassell, E. L. Felix and Russell Desrosiers

ALFALFA Rust (Uromyces [striatus var.] medicaginis), was found affecting one field in Coastal Palm Beach County. About 40 percent of the plants were infected, with severity only light.

BEANS Rust (Uromyces phaseoli [var. typica]) and powdery mildew (Erysiphe polygoni) occurred to a slight extent in the Everglades.

CABBAGE Black leaf spot (Alternaria circinans), was noted lightly infecting a small planting in St. Lucie County. An adjoining row of Brussels sprouts appeared to be infected by the same organism.

CARROT Mosaic (Virus) occurred on 90 percent of the plants in one field. Dodder (Cuscuta sp.) which is so common on the east coast, and rare in the Everglades, was found in the latter area along a good portion of 2 rows, growing on carrots and purslane.

Mosaics were observed in cultivated Queen Anne's Lace.

CELERY Mosaics (Virus) still occur to a slight extent, although most of the celery in this section is now harvested. Cracked stem (boron deficiency) and pencil-stripe (cause unknown) were very severe and occurred on about 75 percent of the plants on 140 acres of new muck which had received no boron. Several spray applications of borax over a period of one month to such celery with extremely brittle and badly

cracked stems resulted in the production of marketable celery.

Early blight (Cercospora apii) became very severe on celery in fields damaged by rain in March. None of the fungicides were fully effective protectants under these circumstances.

CORN Leaf blight (Helminthosporium turcicum) became very prevalent in a large field of hybrid sweet corn, although it did not appear on other corn in the area.

CUCUMBER Downy mildew (Pseudoperonospora cubensis) caused severe damage to 12 acres of cucumbers on sandy soil in the Everglades. Further development appears to have stopped. A trace of mosaic (virus) was observed.

LILY About 75 percent of the Easter Lily plants in 2 small fields showed mosaics (virus).

LESPEDEZA Powdery mildew (Microsphaera diffusa) was found moderately affecting the plants in a mixed field of common and Korean lespedeza in Eastern Palm Beach County.

OKRA Mosaic (virus) occurred to the extent of 60 percent in a small field.

PEAS (BLACK-EYE) A planting in St. Lucie County was moderately affected with powdery mildew (Erysiphe polygoni), and averaged 1 to 2 percent mosaic (virus)-affected plants.

PEPPER A 28-acre field had 85 percent mosaic.

POTATOES A large field of Irish potatoes in Eastern Palm Beach County was 100 percent infected with late blight (Phytophthora infestans), but diligent spraying with Dithane was holding foliage damage to a minimum. Of 3500 acres of potatoes in the Everglades, only a small percentage was lost from late blight, which generally was controlled by Dithane sprays.

OATS Stem rust (Puccinia graminis var. avenae), was present on all plants in a field in Eastern Palm Beach County. Infection was severe.

TOMATOES A survey of commercial tomato fields was made early in April in Eastern St. Lucie County where growers were reporting considerable trouble. All commercial fields visited were affected by several diseases of which late blight (Phytophthora infestans) was the most prevalent and destructive. Moderate to heavy infections of late blight were found especially affecting the foliage with some fruit infection evident. The late blight on tomatoes in the Everglades was checked by warm dry weather in April.

Gray leaf spot (Stemphylium solani) was present in the St. Lucie County fields examined and next to late blight was the most serious disease observed. Some rather heavy stem and petiole infection was present in addition to leaf spotting.

Early blight (Alternaria solani) was more prevalent and severe in the St. Lucie County fields than in any other area of Southern Florida under observation by the writers this winter. Leaf spotting was heavy with some stem lesions present.

Leaf mold (Cladosporium fulvum) was present in the St. Lucie County fields in light to moderate amounts. This disease was first observed on the Coast during the last week of March, and during April has also appeared in several tomato fields in Eastern Palm Beach County.

Blossom-end rot (Physiogenic), has either appeared or increased in most East Coast fields during the month. There has been very little rain and fields, although mostly picked, were suffering from drought and blossom-end rot.

SQUASH A 4-acre field of Cocozelle squash showed 44 percent mosaic.

WHITE DUTCH CLOVER Sooty blotch (Polythrincium trifolii), was seen as a very light infection on 8 to 10 percent of the plants in a field in Eastern Palm Beach County.

EVERGLADES EXPERIMENT STATION
May 1, 1946

GREENHOUSE VEGETABLE DISEASES OF INTEREST IN NEW YORK

A. G. Newhall

Following up the work on control of Botrytis grey mold of leaf lettuce by applications of Thiosan, reported by John S. Niederhauser in PDR August 1, 1945, the writer made three visits a month for five months to seven or eight greenhouses in the Rochester area where approximately 25 acres of winter vegetables are grown under glass, principally, leaf lettuce, tomatoes, and radish, with a little mint, watercress, parsley, and rhubarb. Botrytis was not so prevalent as last winter and control was easily achieved. Results of the experiments will not be given here but a number of diseases encountered are worth mentioning at this time.

The earliest indoor crop of lettuce grown in October-November from seedlings started in outdoor beds sustained nearly a 10 percent loss from virus diseases. There were at least three distinct virus complexes present, including ordinary mosaic, aster yellows, and a necrotic spotting

suggestive of streak, although attempts to transmit the latter by the carborundum method to lettuce, cantaloup, and tomato failed. Growers said they often suffered considerable loss on this first indoor fall crop, seedlings for which are brought in from outdoor seedbeds. In later crops grown entirely inside where regular fumigation for insect control is practiced these diseases are reduced to almost nothing. This was true the past season in three greenhouses under observation.

A small outbreak of lettuce anthracnose (Marssonina panattoniana) was observed on Grand Rapids in one greenhouse in December (1945). This is the first occurrence of this disease in New York in four years. A dozen or so plants were affected. Primary infection may easily have taken place in October. Several plantings were made from the same lot of seed during the season but no other case developed. The fungus could have been brought in from outside as it is known to occur on native Lactuca scariola in the State. Excessive watering, cloudy weather, and low temperatures, mostly in the 40's, favored spread of the fungus which sporulated abundantly. One rather heavy application of a Thiosan dust, containing 50% talc, seemed to check it.

A loss of nearly 25 percent of the fall lettuce crop occurred from Sclerotinia sclerotiorum in a house where the soil had been treated during the previous summer with chloropicrin. The interesting thing in this connection is that leaf lettuce was again planted on this ground bed soon after the first crop was harvested (January). This second crop was cut in March and almost no loss at all was sustained from drop or any other disease. Indications are that the abundant soil moisture prevalent in the fall by reason of the short days, cloudy weather and perhaps too frequent watering were much more conducive to germination of sclerotia than were the longer days, greater sunshine, and drier soil conditions prevailing in the spring.

Loss from downy mildew (Bremia lactucae), which used to be common, was conspicuous by its absence. This is attributed to the fact that for the last six or eight years practically all the lettuce grown is the strain of Grand Rapids known as U.S. No. 1 sent to the writer by Dr. Ivan Jagger and known to be resistant to this fungus. It was of interest, however, that Bremia could be found on the cotyledons and first leaf of a few plants in one or two seedbeds though it never did persist or occur on older leaves.

Circumstantial evidence indicated that the growers who were very liberal with their watering suffered much more from Botrytis and Sclerotinia than those who used less water and more care in distributing it. Both diseases were worse in the late fall than in the spring, a fact attributed to soil moisture and humidity differences. In one greenhouse dusting was begun for control of Botrytis grey mold after plants had been set out in the permanent beds, October 17th. Four applications of a Thiosan-talc (Teresan) 1 to 1 dust were made to 4 plots at 7- or 8-day

intervals. Just before harvest, December 12th, examinations revealed that in the 4 dusted plots of nearly 400 plants each there were but 3 Botrytis- and 3 Sclerotinia-affected plants while in 8 undusted plots adjacent there were 39 Botrytis and 27 Sclerotinia cases. It was concluded that the seedlings, which had been grown in another range, were free from disease, infection having come from the soil of the permanent beds. Observations of the other range throughout the season confirmed this. The dusting program had afforded protection not only against Botrytis but also against Sclerotinia.

The control of buckeye rot of tomato (Phytophthora terrestris) [P. parasitica] apparently was achieved by one grower who, the previous August, applied bordeaux mixture (8-8-50) to the soil at the rate of 1 pint per square foot. He employed over 5000 gallons to a range of 10 greenhouses covering an acre but left a strip 5 ft. wide and 180 ft. long down the side of one house for a check. No rot could be found on the lower clusters of fruit anywhere in the range except on a dozen fruits in this check plot. Phytophthora was isolated on several occasions from these diseased fruits. It is believed from past experience in another greenhouse in the same area that this treatment will keep down the buckeye rot for several years. Other growers are sure that mulching with a strawy manure is an effective way to prevent this rot, and some are investing as much as $10 a thousand square feet in this method of control. Experiments comparing mulch with fungicides are under way.

The control of root knot nematode (Heterodera marioni) is being achieved with volatile fumigants by several growers who have employed "Larvacide", "Iscobrome", or "Dowfume G" applied either with a hand injector or with a two-row subsurface continuous drip applicator. The latter reduced the time and labor of treatment from six days, which was required for steaming, down to four hours in one house, and at a 75 percent reduction in cost. Two comparisons made during the winter between the two methods of applying Iscobrome indicate that best results, by far, can be obtained with the hand applicator. These experiments will be reported elsewhere in more detail.

NEW YORK STATE COLLEGE OF AGRICULTURE, ITHACA, NEW YORK
April 23, 1946

PRELIMINARY EXPERIMENT INDICATES THAT 2,4-D (2,4-DICHLOROPHENOXYACETIC ACID) TREATMENT OF TOMATO MAY REDUCE LOSSES DUE TO FRUIT CRACK MOLDS

W. T. Schroeder and F. G. Smith

Loss of tomato fruit by mold invasion of fruit cracks during the latter part of the season when rains are frequent has concerned a number of canners in New York State. This loss cannot be controlled effectively by

protective fungicide sprays. An experiment designed primarily to determine the effect of several synthetic growth hormones on the ripening of tomato fruit indicated more clearly, however, that applications of 2,4-D reduced the amount of fruit cracks and subsequent mold development. It was originally hoped that such controlled defoliation by hormone sprays might permit the ripening of fruit on spray-protected vines that might otherwise be caught by early autumn frosts. The data below were obtained under only one set of environmental conditions but indicate possibilities that may be of interest to others.

On August 27, several synthetic hormones including 2,4-D at varying concentrations were sprayed on previously non- sprayed tomatoes with every treatment in each of 5 randomized blocks. Before spraying, all fruit showing any red color were removed to prevent any carry-over effect. The first picking on September 9 did not show any significant hastening of ripening among the treatments. The daily mean temperature for the period between application and first picking varied from 62.5° to 79° with a mean of 74.2° F, and the sky was clear on all but 4 of the 13 intervening days.

Rains delayed the next harvest until September 29. During this period, precipitation occurred on 10 days and totaled 7.7 inches. Observations at the second harvest indicated very little cracking of the fruit on the plots sprayed with 2,4-D at a concentration of 1000 ppm. It had previously been observed that this treatment caused the quickest and most complete defoliation.. Counts were made of the total number of fruit, ripe whole fruit, ripe cracked fruit, green fruit, and sunscalded fruit in control plots and those treated with 1000 ppm. 2, 4-D. The data are summarized in Table 1.

Table 1. The effect of 2,4-D on the Per Cent of Fruit Cracks, Sunscalded Fruit, and Ripe Fruit.

Treatment	Total No. fruit	Ripe fruit percent	Sunscald percent	Cracked fruit percent
2,4-D, 1000 ppm.	317	98.4	6.5	9.4
Control	298	92.6	4.1	73.9

The data showed considerably less cracked fruit in the 2,4-D treated plots but little difference in the amount of sunscald or degree of ripeness. Virtually all of the cracked fruit were so overrun by molds as to render them unfit for picking whereas the whole fruit in the 2,4-D sprayed plots were firm and suitable for canning except for a heavy in-

fection of anthracnose. These plots, however, had not received any fungicide application and the anthracnose was equally severe in all plots regardless of treatment. It is suggested that 2,4-D treatment may prove useful in controlling losses due to fruit cracks, especially in wet seasons, and that further investigation is warranted.

NEW YORK STATE AGRICULTURAL EXPERIMENT STATION
GENEVA, NEW YORK, April 22, 1946

SEED PROTECTANTS ON SWEET CORN IN RELATION TO PLANT VIGOR

B. H. Davis and C. H. Haenseler

In summarizing the results of the 1943 national seed protectant tests on sweet corn, Haenseler (PDR Suppl. 145, page 7) states that weak seedling counts made in 13 tests showed the largest total from untreated seed and the lowest total from the .375% Arasan treatment, which gave the highest total emergence. Haenseler concludes that "greater benefits may be derived from seed treatments than are shown in the 'total emergence' figures ordinarily used to evaluate seed protectants". The data on weak seedling counts from 19 locations in the 1944 national tests likewise showed that without exception the higher the mean emergence for each protectant used the lower the percentage of weak seedlings (PDR Suppl. 161, p. 53). In these tests the individual cooperators recorded the number of obviously weak seedlings along with the total emergence but made no determinations on the mean plant height for the various protectants with the exception of one test reported by Brooks in Florida. Data from this one test (PDR Suppl. 161, p. 58), based on 4 field measurements per plot, showed that increases in mean plant heights were associated with increased stands.

In 1945 an experiment was conducted at the New Jersey Agricultural Experiment Station in order to get further data on the relationship between emergence counts and plant vigor from treated and untreated seed. A lot of treated seed held over from the 1943 national tests was planted on May 29th, 1945. Each treatment and the control was replicated 5 times with 100 seed in each replication. On June 15th emergence counts were made and on the 26th all plants were pulled, counted and measured. The distance between the first node and the tip of the longest leaf was considered as the plant height. Treatments, application rates, and data obtained are recorded in Table 1.

The data in Table 1 show significant increases from all treated lots in seedling emergence, in percentage of strong plants and with one exception (Semesan Jr.) in mean plant heights. Differences among the several treated lots are not statistically significant except between Arasan (3 oz rate) and Semesan Jr., the former giving an increase in mean plant height.

Table 1. Effect of sweet corn seed protectants on emergence and seedling vigor.

Treatment		Plants per 100 Seed			Mean Height June 26		
Protectant	Rate per bu.	Total June 15	Total June 26	Strong* Plants June 26	All plants	All strong* plants	Best 50 plants
	oz.				inches	inches	inches
Semesan Jr.	1.5	86.6	87.0	80.8	15.62	16.12	18.06
Arasan	1.5	89.2	88.4	85.2	16.92	17.26	19.30
Arasan	3.0	88.0	88.4	87.0	17.66	17.80	19.68
Spergon	1.5	87.2	87.6	84.4	17.24	17.76	19.48
Spergon	3.0	85.8	86.2	81.0	16.18	16.68	18.72
Check		79.6	81.2	69.6	14.12	14.98	16.34
L.S.D. 5% point		4.4	5.0	6.7	1.81	1.65	1.94

* All plants 10" in height were considered strong plants.

These data show that the increased stands obtained with sweet corn seed protectants are accompanied by a decrease in the percentage of weak seedlings and by an increase in the average plant vigor.

NEW JERSEY AGRICULTURAL EXPERIMENT STATION

REPORTS ON TOBACCO BLUE MOLD

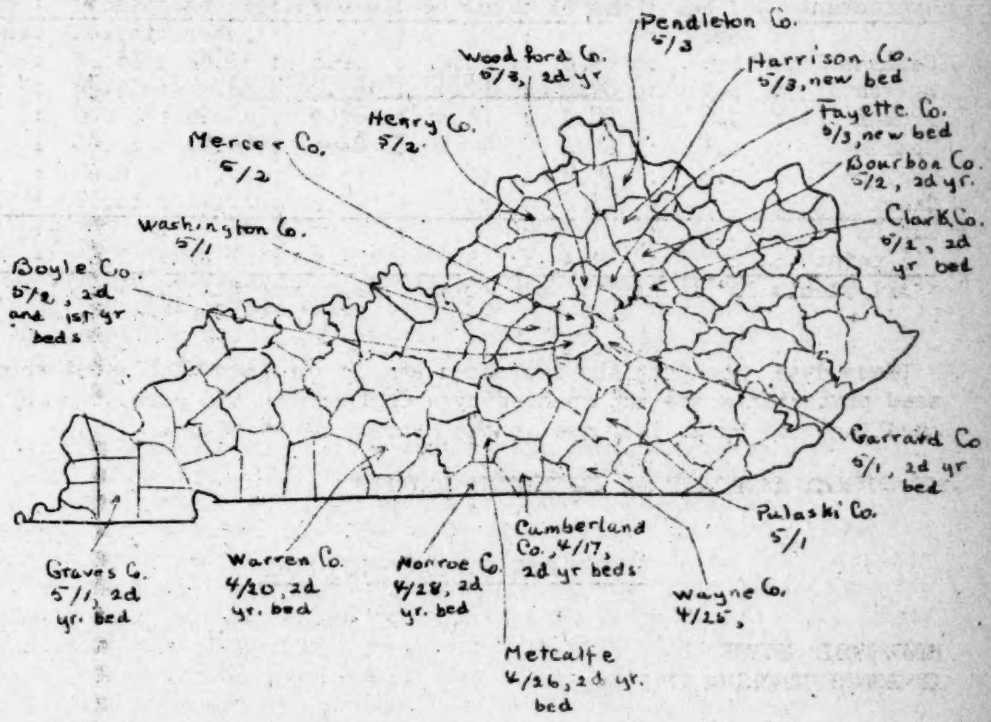
BLUE MOLD SEVERE IN SOUTH CAROLINA IN 1946

By T. W. Graham

Tobacco blue mold [Peronospora tabacina] was very destructive in South Carolina in 1946. The disease appeared unusually early (about March 10) and weather conditions were favorable for its spread and development for a period of five weeks. In some communities plant mortality was high. In Florence and Darlington Counties 50 to 75 percent of the plants in many beds were killed by blue mold. In areas where fewer plants were actually killed defoliation was severe and transplanting was delayed two to three weeks. As a consequence of the blue mold outbreak many growers will transplant very late or obtain plants from growers who treated their beds to control the disease. The control method used most extensively was Fermate dust. Growers who used this treatment had excellent results and were able to transplant their crops at the normal time. Approximately 20 tons of Fermate dust (15% Fermate in Pyrophyllite) were used by tobacco growers in South Carolina this season.--Agent, Division Tobacco, Medicinal & Special Crops, U.S. Bureau of Plant Industry, Soils and Agricultural Engineering, Florence, South Carolina. April 20, 1946.

BLUE MOLD IN KENTUCKY

By W. D. Valleau


Pendleton Co. 5/3
Woodford Co. 5/3, 2d yr
Harrison Co. 5/3, new bed
Fayette Co. 5/3, new bed
Henry Co. 5/2
Mercer Co. 5/2
Bourbon Co. 5/2, 2d yr.
Clark Co. 5/2, 2d yr bed
Washington Co. 5/1
Boyle Co. 5/2, 2d and 1st yr beds
Garrard Co 5/1, 2d yr bed
Pulaski Co. 5/1
Cumberland Co. 4/17, 2d yr beds
Graves Co. 5/1, 2d yr. bed
Warren Co. 4/20, 2d yr. bed
Monroe Co 4/28, 2d yr. bed
Wayne Co. 4/25,
Metcalfe 4/26, 2d yr. bed

On April 17 blue mold was found in Cumberland County in two beds used last year and again this year. The plants were about 1 inch across when first affected. New beds seemed free at this time. Since then the disease has been reported as shown as on the accompanying map. There seems to have been no "blow-in" from Georgia. Infection originated mostly from inoculum carried over in second-year beds.

Weather was dry for nearly a month, until the beginning of May.--Kentucky Agricultural Experiment Station.

BLUE MOLD IN SOUTHERN MARYLAND

By E. A. Walker

I visited the tobacco plant beds with Mr. Julius Johson on April 12, and found blue mold on plants averaging in size from a dime to a nickel in one plant bed. -- University of Maryland, April 15,

PEACH WART FOUND IN CALIFORNIA

G. L. Stout and R. L. McClain

On June 5, 1945, while examining peach trees for peach mosaic at Yucaipa in San Bernardino County, California, green peach fruits showing typical peach wart as described by Blodgett (Phytopath. 33 (1): 21-32. 1943) were seen by the writers on peach trees of the Candoka variety. This is the first time peach wart has been found and recognized in California. Specimens were shown to L. C. Cochran, of the U.S.Department of Agriculture, who agreed as to the diagnosis.

The orchard manager, who was present at the time and who called our attention to the warty fruit with an inquiry as to its cause, told us that the condition was present on a number of his Candoka trees, not on his other varieties, and that it first had been noted on the Candoka trees during the previous season (1944) when it had caused a considerable number of cull fruits. Prior to 1944 little or no fruit had been produced on the affected trees. History indicated that approximately 285 peach trees on the property had been topworked to the Candoka variety in the spring of 1941. Records of the local county agricultural commissioner, and also statements by the nursery agent who sold the scion wood to the orchard owners, showed the scion wood to have been received from a nursery near Wenatchee, Washington, in February, 1941. The local county agricultural commissioner also has a record of 15 Candoka peach trees having been received by these same orchard owners in February 1941, from the same source.

The Candoka trees were located on a ranch with a large acreage of peach trees of other varieties. A survey of all peach trees on the property was made by one of the writers (McClain) and the agricultural commissioner and members of the commissioner's staff. Peach wart was found only on trees of the Candoka variety, and of approximately 300 trees of this variety, warty fruit was found on 203 of them. Since the manager previously had instructed the fruit thinners to remove warty fruit in thinning, most likely additional trees had shown some warty fruit earlier in the season. Indications were that the disease had been brought onto the property in the scion wood of the Candoka variety, and possibly also in some or all of the 15 Candoka nursery trees, from Wenatchee. The owners

of the orchard were persuaded to remove all trees which were of the Candoka variety or which had been grafted with that variety.

Through the assistance of various county agricultural commissioners in the State, other scattered plantings of the Candoka variety were located and inspected in several counties. Most of these plantings contained a few to 100 trees, and none over 500 to 600 trees. Inspections of such plantings conducted by R. L. McClain in Southern California and R. C. Baines in other parts of the State did not reveal any additional cases of wart except in eight Candoka trees on four properties in the Banning Heights area of Riverside County. These four properties contained 1517 Candoka trees.

All properties containing the wart infected trees were located in the same general area within a few miles of each other and all wart trees have been removed. These properties will be carefully watched in the future for any additional wart trees.

BUREAU OF PLANT PATHOLOGY, CALIFORNIA STATE DEPARTMENT OF AGRICULTURE, SACRAMENTO, CALIFORNIA

SOUR CHERRIES IN VIRGINIA KILLED AS A RESULT OF 1945 LEAF SPOT EPIPHYTOTIC

A. B. Groves

A number of instances have been observed in Northern Virginia where previously healthy sour cherry trees have died since last year and for which no plausible explanation other than that of exhaustion following the 1945 leaf spot [Coccomyces hiemalis] epiphytotic seemed apparent. Confirmation of this diagnosis was afforded by a block of young cherry trees used in experimental spraying last year, and where the unsprayed checks were distributed as entire rows at intervals through the block. When observed at full bloom this year (April 19) the check rows were devoid of bloom or active leaf buds whereas all sprayed blocks were in full and abundant bloom. The branches had not yet become brittle, but shallow cuts through the outer bark revealed it to be brown, obviously dying. Two six-year-old and previously well-cared-for orchards near Winchester were completely killed as a result of the 1945 defoliation. In both instances the owner had left the trees unsprayed after the crop had been destroyed by frost.

Briefly reviewing the 1945 season, leaf spot infection was apparent by the first of May at Winchester, and as a result of very favorable weather conditions infection built up rapidly, defoliation was beginning by mid-May and all but tufts of terminal leaves had fallen within a month. The trees continued growth for perhaps another month but infection and defoliation progressed as rapidly as growth, the trees remaining

bare the rest of the season upon cessation of growth. It seems significant to note that trees receiving even the poorest spray treatment, which delayed defoliation about five weeks, survived the winter in good condition and have now set a good crop.

It is now apparent that many hundreds of sour cherry trees have been lost in Virginia as a result of exhaustion following the 1945 leaf spot epiphytotic which began early and persisted all season. Even poor spray treatments were adequate to permit the tree to build up enough strength for survival in apparent good condition. Winter injury does not appear to have been a factor in killing the trees. A combination of two circumstances, a freeze-out of the crop in many orchards with consequent abandonment of spray programs together with the worst leaf spot epiphytotic on record in Virginia, were the major causal factors in this loss.

VIRGINIA AGRICULTURAL EXPERIMENT STATION"

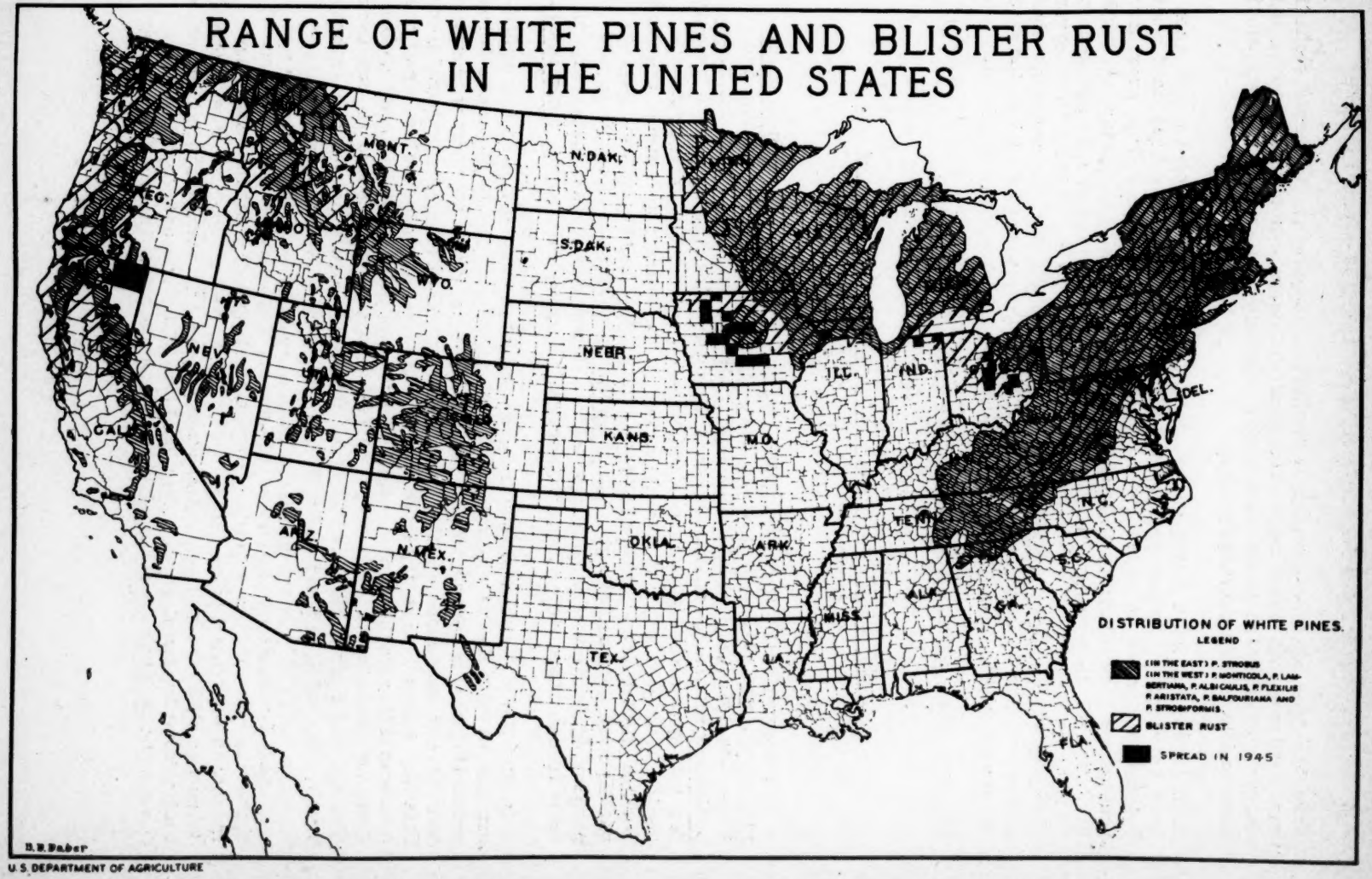
SPREAD OF WHITE PINE BLISTER RUST DURING 1945

James F. Martin

In northern California the known range of blister rust [Cronartium ribicola], was extended eastward into the southern Warner Mountains, Modoc County, where the disease was found for the first time on Ribes petiolare within the Modoc National Forest. Climatic factors necessary for the spread and intensification of the rust in the Pacific Coast region were generally unfavorable, but in the locality between the East Fork of Indian Creek and the Slater Butte Road in the western part of the Klamath National Forest, R. sanguineum bushes had from 50 to 100 percent of their leaves infected with blister rust, while elsewhere on this forest ribes infection was generally light, even on R. sanguineum.

In the Sierra Nevada numerous infected ribes were found at the southern end of the Shasta and the northern end of the Lassen National Forests, where the rust appeared to have intensified on ribes to a greater extent than it had elsewhere in either the southern Cascades or northern Sierra Nevada. Ribes leaves infected with blister rust were obtained from the southern Eldorado National Forest for the second consecutive year, and four new pine infection centers of 1938 origin were located on the Lassen, Plumas, and Shasta National Forests within the previously known infected areas. The pine infection found at these places was destroyed.

In the Northwestern region no blister rust infection was found on white pine or ribes beyond its previously known distribution. Heavy infection was found on the leaves of six R. petiolare bushes on Clematis Gulch and Glen Creek in the Mammoth area, in the northwestern portion of Yellowstone National Park. This confirms the original report of the disease on R. petiolare leaves from Clematis Gulch in 1944 and may be


RANGE OF WHITE PINES AND BLISTER RUST
IN THE UNITED STATES
MONT.
N.DAK.
S.DAK.
WYO.
NEBR.
NEV.
CAL.
KANS.
ARIZ.
N.MEX.
OKLA.
TEX.
ILL.
IND.
MO.
ARK.
MISS.
LA.
TENN.
ALA.
GA.
S.C.
N.C.
VA.
DEL.
FLA.
DISTRIBUTION OF WHITE PINES.
LEGEND
(IN THE EAST) P. STROBUS
(IN THE WEST) P. MONTICOLA, P. LAMBERTIANA, P. ALBICAULIS, P. FLEXILIS P. ARISTATA, P. BALFOURIANA AND P. STROBIFORMIS.
BLISTER RUST
SPREAD IN 1945
H.R. Baker
U.S. DEPARTMENT OF AGRICULTURE

indicative of undiscovered infected white pine in that general locality. Blister rust infection has been distributed generally throughout the Northwestern region since 1937, and severe damage to pine occurred in unprotected young stands in 1941. Since then there has been no serious general build-up of the disease on white pine. Weather conditions in the spring of 1945 were ideal for spread of the rust from pine to ribes, but the heavy initial infection of ribes was largely dissipated by a long period of dry, hot weather continuing from late June to late August. This kept the spread of the disease from ribes to pines from developing into the wavelike proportions observed in 1937 and 1941.

In the North Central region ribes infection was found for the first time in 22 counties in 4 States, as follows: Carrol County, Illinois; Elkhart and Steuben Counties, Indiana; Buena Vista, Carrol, Clay, Dallas, Emmet, Greene, Hamilton, Hardin, Humboldt, Mahaska, Marion, and Warren Counties, Iowa; and Columbiana, Crawford, Delaware, Franklin, Morrow, Muskingum, and Perry Counties, Ohio. Infected white pines were found for the first time in Crawford and Ogemaw Counties, Michigan. As in 1944, the greatest extension of the rust into new areas occurred in Iowa, where infected ribes were found in 12 new counties. Considerable variation in the intensity of rust development on ribes was noticeable. In Illinois, Indiana, Ohio, and the lower portions of Michigan and Wisconsin ribes infection was light and relatively scarce, but in Iowa, Minnesota, and the upper portions of Wisconsin and Michigan ribes infection was normal. The amount of infection on white pine has increased considerably in northern portions of the region during recent years, and numerous cankers of 1941 and 1942 origins were observed in 1945.

In the Southern Appalachian region two centers of infected white pines were discovered in Grayson County, Virginia. These were the first found in southwestern Virginia and at the time were the most southern-known pine infection centers in the region. Late in the summer, however, a small area of diseased white pine was discovered near the headwaters of New River in northwestern Ashe County, North Carolina. This is the first area of infected white pines found in North Carolina. It is about three acres in extent, and the infection appears to be of 1936 origin. The stand was not considered of sufficient value to be included with areas being protected by ribes eradication. It averages about 75 pines per acre and these trees seeded in naturally from one old tree. The pines are uneven-aged, mostly under 20 years old, and Ribes cynosbati is generally distributed. About half of the trees are estimated to be infected with blister rust. Cankers are present on stems and branches, and some of the pines already have been killed by the disease. Blister rust infection was reported for the first time from Anne Arundel and Cecil Counties, Maryland, on cultivated ribes.

In the Northeastern region blister rust is generally distributed, and ribes eradication has been progressively extended within the white pine area over a period of many years. Control of the disease has been estab-

blished in those parts of the worked area on which these bushels have been so reduced, as a result of ribes eradication, that a serious rust hazard to the pines no longer exists. Continued suppression of ribes will keep them on the decline and maintain effective control of the disease.

The spread of the rust from ribes to pines has continued in unprotected areas, as well as in those parts of the protected areas where these bushes have not been kept sufficiently suppressed to control the disease. Spread in these protected areas is due to unavoidable delays in scheduled reworkings and to logging and other disturbances which bring about the germination of viable seeds that have accumulated in the duff of the forest florr. In general, the prevailing effectiveness of the ribes-eradication work in controlling the disease is shown by the relative scarcity of new cankers in protected areas and the presence of white pine reproduction free of blister rust infection.

Most of the younger pines infected prior to initial control work have been killed by the rust and gradually disappeared from the stands. Meanwhile, damage resulting from early infection of the older and larger pines, which are killed more slowly by the disease, has become increasingly apparent. This is manifest in many stands by a growing number of dead and dying trees in the older age classes. Damage of this sort is most prevalent in Warren and Essex Counties, New York, the upper Connecticut River Valley in New Hampshire and Vermont, and in most sections of Maine outside of York and Cumberland Counties.

DIVISION OF PLANT DISEASE CONTROL
BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE
February 26, 1946

DIE-BACK AND STEM CANKER OF CAMELLIA

Douglas C. Bain

Die-back of Camellia was briefly described in 1934, by Post (2), and Gloeosporium was reported to be associated with the disease. West (3), in 1941, reported die-back of Camellia in Florida. Species of Diplodia and Phomopsis, isolated from diseased material, did not infect plants that were inoculated. Later (4), the same author reported that in addition to Diplodia and Phomopsis, a species of Gloeosporium was isolated. He indicated that inoculation experiments involving all three fungi were in progress. In 1942, Davis and Pirone (1) described a stem canker of Camellia and established the causal organisms as Glomerella cingulata. Surgery and sanitary measures were recommended for control, and white-flowered varieties were indicated as being more susceptible to the disease. None of these notes completely and adequately describes the disease as it occurs in Mississippi nor offers

a satisfactory control measure. Other reports of the occurrence of Gloeosporium and G. cingulata on Camellia have appeared in the literature from time to time.

After a brief surveys of nurseries in February, particularly Camellia nurseries along the Gulf Coast area, it became apparent that the disease was prevalent and was serious. As a matter of fact, some growers were frankly alarmed, especailly so since they did not understand the disease and had no method of control. The facts that a considerable amount of money is involved in the culture and sale of Camellia and that the disease has inflicted either total loss of plants or otherwise caused serious damage are reasons enough to re-examine the work done and, if possible, add to it.

The most obvious symptom of the disease shows up in the young shoots during the early spring season. Apparently after infection takes place the subtending leaf is abscised and shortly thereafter the basal portion of the shoot begins to turn black. This discoloration progresses distally and is followed by a wilting, at first of the terminal leaves and later of all the leaves. Leaf wilting is also accompanied by wilting of the shoot. Often one or more of the leaves turn black. Under conditions of ample moisture, acervuli of a species of Gloeosporium appear at first at the basal end of the shoot and later may be formed all over the shoot. Dead shoots apparently remain on the branches indefinitely. In the early stages of the disease, tangential sections of the branch and its shoot reveal a brownish discoloration which extends from the base of the shoot through the nodes and into the branch above and below the node. This disease is termed "die-back", and appears to be the initial stage of stem canker, described below.

Cankers have been observed only at nodes of stems or branches, but not on current season shoots. In general, the cankers are ovate-elliptic to almost circular, and surround a dead branch or its stub. The width and length are variable but are usually under 2 cm. In many instances, however, cankers will completely girdle the stem or branch and frequently death of the distal portion is the result. Ordinarily, the cankers are only 2-3 mm deep but penetration to the pith has been observed. Cankers are considerably less conspicuous than die-back.

Tissue platings of both die-back and canker have consistently yielded one or more species of Gloeosporium. Inoculation experiments carried out thus far clearly show that some of the isolates can reproduce die-back symptoms as they occur in nature. Typical symptoms of the disease begin to show up in shoots a week to ten days following inoculation by the needle prick method. Thus far, only current season shoots have been inoculated and it will perhaps be some time before the connection of die-back and canker can be definitely established.

Through the courtesy of Mr. Fred P. Moreton, Ocean Springs, Mississippi,

a spray program has been started in an effort to control the disease. Four well-known fungicides are being used at 10- and 20-day intervals. The schedule was started on April 1, shortly after this year's set of shoots was well out. There are indications, however, that the schedule was started too late in the season, since a considerable amount of die-back has been observed on all sprayed plants thus far. These results, plus the fact that infection invariably manifests itself at the base of the shoot, more or less fit in with the writer's idea that infection takes place at or about the bud scale scars shortly after the buds begin to swell early in the season. If this is true, then spraying should be started shortly before the buds begin to swell and should be continued until the shoots are out and have passed their tender stages. Of course, in considering control measures, sanitary practices should not be overlooked.

According to observations made along the Gulf Coast this spring, the colored varieties are just as susceptible as the white varieties. Die-back and canker have been observed on the following varieties growing at Ocean Springs: Purple Dawn, Adolph Audusson, Marchioness of Exeter, Glen Forty, Arpacia, Magnoliae Flora, Lady DeVere, Annabalis, and Alba Plena. Magnoliae Flora and Alba Plena appear to be considerably more susceptible than the other varieties listed; on the other hand no die-back or canker was observed in the varieties Prof. Sargent and Perfection Alba, which indicates these are resistant. Both of these varieties were growing in among others which were heavily infected.

It is of interest to note that some of the popular writings on Camellia culture and gardening attribute the disease to "Phomopsis blight" or poor soil conditions. They either recommend spraying (with no particular schedule) for "perfect control" or treatment of soil to correct drainage or to lower the pH value. On the Mississippi Coast, the indications are that soil conditions have no effect on the incidence or severity of the disease.

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AGRICULTURAL EXPERIMENT STATION, STATE COLLEGE, MISSISSIPPI

A DISEASE OF ENGLISH IVY IN GEORGIA

Kenneth H. Garren

In February of this year our attention was called to a disease of ground plantings of English ivy (Hedera helix L.) in Atlanta where the disease is reported as being widespread. Since that time it has been observed in one planting in Griffin. The disease as found in Georgia appears to be the same as that described by Weiss from the vicinity of Washington, D. C. (PDR 26: 331-332. 1942).

In the ivy beds observed plants had died in scattered patches of one to two feet in diameter. Some of the patches had coalesced, producing large irregular areas of dead plants. There was no evidence of winter injury, or scale insects, and soil analysis revealed no mineral deficiencies.[1] In all plantings observed the beds were shaded for a considerable portion of the day. Examination of living plants adjacent to the dead plants showed that necrotic lesions were present on many of the stems. Necrotic areas were found also on the majority of the leaves. Infrequently petioles were cankered. The diseased areas on the leaves varied in size from minute to as large as one-half of the leaf and appeared to have spread inward from the outer edge of the blade.

Colletotrichum trichellum (Fr.) Duke found on the surface of stems of dead plants and in some of the lesions on stems of living plants. This fungus developed abundantly on leaves, stems, and petioles when diseased plants were kept in a moist chamber from three to four days. Occasionally leaves with necrotic spots were found in beds of ivy the plants of which otherwise showed no evidence of disease. C. trichellum also developed readily on these leaves when they were kept in a moist chamber.

Since Weiss did not attempt to isolate the organisms associated with the disease, isolations were made from material collected in Atlanta and Griffin. Diseased tissue taken from living plants was surface sterilized with 1:1000 mercuric chloride and plated on 2% tap-water agar. Although C. trichellum was isolated from diseased tissue of leaves and petioles, it could not be isolated from stems. Furthermore, it did not appear in the majority of the isolations. The organism which appeared most frequently in isolations from leaves and petioles was a Phyllosticta sp. Isolations from stems were about equally divided among Fusarium spp., a Diplodia sp. (also isolated from roots), and a Phoma sp. Two bacteria, an Alternaria, and the usual variety of molds and sterile fungi, were isolated from all plant parts. Among the fungi isolated only C. trichellum could be readily identified in culture. The genera to which other fungi belong could be determined from cultural characteristics. These fungi could not, however, be placed in any species of those genera described from ivy.

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[1] Entomological examinations and soil analysis by Theodore L. Bissell and L. C. Olson, respectively, of the Georgia Experiment Station.

C. trichellum has been reported as causing severe injury to ivy in Italy (Rev. Appl. Mycol. 13: 309, 1933). The review of this report leaves the impression that the disease was confined largely, if not entirely, to greenhouse plantings. In another study of a greenhouse disease of ivy, C. trichellum and Phyllosticta hedericola Dur. & Mont. were found to be secondary invaders of leaves following severe attack by Bacterium hederae Arnaud (White and McCulloch, Jour. Agr. Res. 48: 807-815. 1943). Lesions observed in Georgia are similar to those illustrated in the latter report.

In view of the scarcity of information on diseases of outdoor plantings of ivy, inoculation tests are planned with the bacteria and all fungi isolated.

GEORGIA AGRICULTURAL EXPERIMENT STATION
EXPERIMENT, GEORGIA

AN EPIPHYTOTIC OF VERTICILLIUM WILT IN SOUTHERN CALIFORNIA

Kenneth F. Baker and Warren F. Locke

The unusually cool and cloudy summer of 1944 in coastal southern California favored an outbreak of Verticillium wilt on annual crops, particularly in the Arroyo Grande and Guadalupe-Santa Maria areas. The facts are here reported and discussed in relation to published data on epidemiology of the disease.

The disease was prevalent in flower seed fields near Arroyo Grande and Guadalupe by July, and produced heavy losses by August. Verticillium albo-atrum R. and B. was cultured from the following plants taken from these fields: several varieties of Antirrhinum majus, the fields of which were severely damaged; Centaurea imperialis[1]; several varieties of C. cyanus, fields of which were moderately injured: Helichrysum bracteatum, which was severely damaged; several varieties of Petunia hybrida[1]; Salpiglossia sinuata, fields of which were severely injured; Salvia farinacea[1]; Chrysanthemum maximum var. Shasta Daisy[1]; Schizanthus pinnatus[1]; Celosia argentea var. cristata[1] in a field near Buellton.

The mean monthly air temperature (°F.) and departure from normal for the nearby San Luis Obispo and Santa Maria weather stations, respectively, were as follows:[2]

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1 Apparently new host records for the fungus.
2 Compiled from Climatological Data, California Section, U.S.Dept. Commerce, Weather Bureau. Normal is the 48-year average.

January	52.6	+0.7	51.2	+1.1
February	50.7	-3.0	50.6	-0.8
March	56.8	+1.8	55.0	+1.4
April	53.5	-3.2	53.8	-2.0
May	57.2	-1.2	58.0	-0.9
June	60.8	-1.2	60.6	-1.0
July	63.6	-0.9	61.8	-1.7
August	64.0	-0.8	60.8	-3.1
September	64.6	0	62.1	-1.2
October	62.1	-0.2	60.1	-1.2

For the months February through September, when crops were in the fields, the average departure from normal was -1.06° F for San Luis Obispo and -1.16° F for Santa Maria.

Studies (1, 2, 3, 4; and others) on the soil temperature relations for Verticillium wilt give about 54° F as the minimum, 70-75° F as the optimum (with little wilt appearing above 77° F), and 84° F as the maximum. The temperature range for wilt production was found (2) to be broader (64-84° F) at the 63 percent of water-holding capacity than at 45 percent (71-81° F). While the soil temperatures of the Santa Maria section in 1944 are unknown, it seems certain from air temperature records that they must have been below the reported optimum for the disease. Since the summer of 1944 was decidedly cooler than the usual season in which wilt is scattering and unimportant, it appears improbable that the epiphytotic occurred because of the low temperatures of that season.

Monthly precipitation and departure from normal for San Luis Obispo and Santa Maria, respectively, were as follows:[2]

January	1.77	-3.42	1.32	-2.00
February	9.45	+5.30	4.69	+1.94
March	2.61	-0.93	1.36	-1.17
April	2.22	+1.03	2.46	+1.59
May	0.24	-0.49	0.11	-0.43
June	0.04	-0.05	0.01	-0.01
July	Trace	0	0	0
August	0	-0.04	0	-0.06
September	0	-0.28	0	-0.24
October	0.14	-0.62	0.12	-0.49

For the months February through September, there was an accumulated excess of 4.54 inches for San Luis Obispo and 1.62 inches for Santa Maria. Because furrow irrigation is practiced in the area, a high soil moisture is less dependent on rainfall than in areas such as the Niagara Peninsula (2), where uniformly distributed summer rainfall apparently determines the incidence of epiphytotics. It would seem probable that the occurrence of an epiphytotic on annual crops in coastal California would be little affected by rainfall. However, the uniformity of soil moisture

would be affected to some extent by the frequency of cloudy, humid weather.

The number of overcast (cloudy and partly cloudy) days and the departure from the average of the previous 10 years for San Luis Obispo and Santa Maria, respectively, were as follows:[2]

January	18	+3.9	19	+5.0
February	16	+0.2	15	+0.3
March	6	- 7.9	10	+2.7
April	9	+1.5	16	+4.8
May	18	+10.7	24	+17.1
June	18	+13.6	18	+12.3
July	17	+14.1	21	+15.7
August	7	+4.7	8	+1.4
September	12	+8.7	17	+11.2
October	5	+1.9	17	+8.0

During the months February through September, there was an average excess of 5.7 days of cloudy weather for San Luis Obispo and 8.2 days for Santa Maria; the percentages of days which were overcast were 42.4 and 53.1, respectively. In the previous 10 years at San Luis Obispo the largest total number of overcast days during these 8 months was 71 in 1938, compared with 103 in 1944; at Santa Maria the greatest number was 108 in 1940, compared to 129 in 1944. From these data it would seem probable that the soil moisture in 1944 was more uniform and higher than usual. Such a condition might have enabled (2) the fungus to produce disease at lower soil temperatures than usual, and thus produce the outbreak of 1944.

Fields that had serious wilt losses in 1944 had practically no disease in 1945. This was particularly striking in a field of Helichrysum that had heavy losses in 1944. Much seed shattered and produced a disease-free volunteer crop in 1945 in the identical area of the previous loss. This is in line with observations of others (2,4) and with common field experience in California. There is a stronger tendency for Verticillium than for Fusarium wilts to occur in epiphytotics, as would be expected on the basis of the requirements of the physical environment for each. The severity of Fusarium wilts largely is determined by soil temperature, whereas Verticillium wilt is affected critically by soil temperature and moisture.

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BRIEF NOTES ON PLANT DISEASES

SWEET POTATO INTERNAL CORK IN GEORGIA

By Julian H. Miller

During the past February and March, the writer has completed a survey of sweet potato houses and hills. Internal cork was found in the following counties: Appling, Carroll, Clarke, Dodge, Dooly, Floyd, Polk, Spalding, Thomson, and Tift. In cutting the potatoes to determine the presence of the disease, approximately fifteen were used in each case. This internal cork varied in appearance from very small, black, necrotic spots which would hardly be noticeable to large areas, one to three inches in length and about one-half inch in diameter. In the latter cases, the overlying parts of the potatoes were shrunken and so one could guess at this by an outside observation. All of the seed stock from which these potatoes were grown had the internal cork last and year and all of them were from two seed sources except one case in Polk County. No cork was found in locally grown potatoes that had been in Georgia for five years or more. This disease was found in potatoes grown on soil types varying from red Cecil clay to Norfolk sandy loam. During the survey, other stocks of potatoes were examined on the same or adjoining farms and no cork was found in those coming from the two chief sources.--Department of Plant Pathology, University of Georgia

A FIELD TEST OF LATE BLIGHT RESISTANCE IN TOMATOES

By C. F. Andrus and B. L. Wade

The epidemic of late blight (*Phytophthora infestans*) on tomatoes in south Florida, noted previously by Harrison[1], carried over into the winter crop and provided an exceptional opportunity for a field test of resistance. All of the relatively few commercial varieties observed were very susceptible. Among 15 advanced breeding lines from the U.S.Regional Vegetable Breeding Laboratory under propagation at Homestead, 3 were highly resistant to late blight.

The Homestead planting of the Vegetable Breeding Laboratory lines was made possible through the cooperation of W. D. Moore, U. S. Department of Agriculture, Ft. Lauderdale, and G. D. Ruehle and H. I. Borders, Florida Agricultural Experiment Station, Homestead. The plants were set in the field in January. Drs. Moore and Borders examined the field on March 25th and reported the 3 lines to be highly resistant to late blight. The writers examined the tomatoes on April 8th, accompanied by

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[1] A. L. Harrison, Late blight of tomatoes on west coast of Florida. USDA, Pl. Dis. Reptr. 30: 49-50. 1946.

Drs. Moore and Borders and Dr. A. L. Harrison, pathologist of the Bradenton Vegetable Crops Laboratory.

The this date the late blight epidemic had subsided, but there were still many active lesions and the effects of the earlier attack were still very evident. In the minds of the five persons who examined these tomatoes on April 8th there was no doubt that the 3 lines referred to possessed a very high degree of resistance to late blight. The foliage appeared to be entirely free but an occasional fruit showed lesions. In the susceptible lines, which included Rutgers, a high percentage of the fruits were badly damaged by the late blight fungus.

All of the 3 late-blight-resistant lines possess near-immunity to Fusarium wilt, and one of them is resistant also to collar rot (Alternaria) and early blight (Alternaria). This latter tomato had previously been entered in southern field trials under the designation "STEP 22" and can be seen this summer at various field stations in the South.

All of these tomatoes have in their ancestry Lycopersicon pimpinellifolium but are of commercial size.--U. S. Dept. Agr. Regional Vegetable Breeding Laboratory, Charleston, S. C.

GERMINATION OF TOBACCO SEED PREVENTED BY CYANAMID

By E. M. Johnson and W. D. Valleau

A number of tobacco growers in Jessamine County reported that tobacco seed sowed early in March had failed to germinate by early April. A second sowing about the second week in April failed to germinate despite favorable conditions. Upon investigation it was found that all of the growers who reported failures used one brand of a mixed fertilizer purchased from the same dealer. On the surface of the soil of the seed beds, where the tobacco seed failed to come up, there were numerous black spherical pellets 1/16 inch in diameter. It was suspected that these were calcium cyanamid. A sample was collected from the fertilizer remaining from the lot used on one of the tobacco beds. An analysis by the Department of Feeds and Fertilizers showed that this fertilizer contained over five percent calcium cyanamid. Most of the growers mixed the tobacco seed with this fertilizer for ease of sowing. The rate of application to the soil was about 30 to 100 pounds of mixed fertilizer or 7 1/2 to 25 pounds of calcium cyanamid per 100 square yards.

On tobacco beds treated with this fertilizer, containing calcium cyanamid, the few weeds present mostly along the margins were either much yellowed or partially withered. Knotweed, narrow-leaf plantain, and cheesemallow plants had necrotic lesions, one half to one inch long, on the stem below the soil surface. Below this depth, apparently in soil free of fertilizer, the roots were normal. Small crabgrass plants

were killed by the fertilizer.

It was reported that one grower used this fertilizer to top dress a winter cover crop of vetch and rye. All of the vetch was killed and the rye was damaged. Another farmer reported that he killed radishes, lettuce, and tomato plants with the fertilizer.--Kentucky Agricultural Experiment Station.

APRIL WEATHER

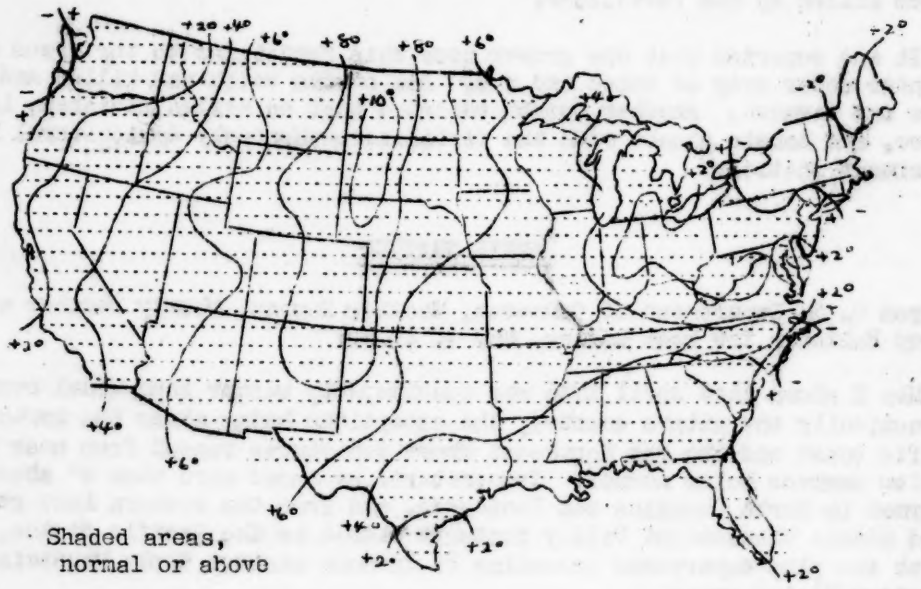
(From U. S. Department of Commerce, Weather Bureau, Weekly Weather and Crop Bulletin for week ending, May 7, 1946.)

Map I shows that April 1946 was considerably warmer than usual over practically the entire country, the exceptions being along the north Pacific coast and the far Northeast where departures ranged from near to a few degrees below normal. Temperatures averaged more than 4° above normal in North Carolina and Tennessee, and from the western Lake region and middle Mississippi Valley westward almost to the Pacific States, with the plus departures exceeding 8° in many eastern Rocky Mountain and western Plains areas.

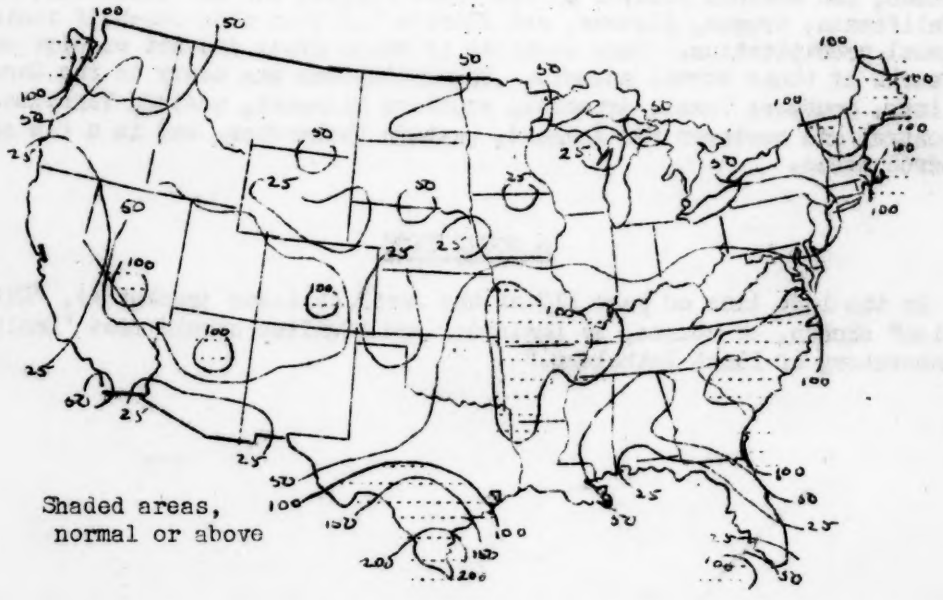
Map II presents the areal distribution of total precipitation for April 1946. Most northern sections of the country from New Jersey to Idaho, the western portion of the Great Plains, the far Southwest, and California, Oregon, Alabama, and Florida had less than one-half their usual precipitation. Many stations in these areas did not receive one-fourth of their normal amounts. Precipitations was heavy in the Carolinas, southern Texas, Arkansas, southern Missouri, western Tennessee, central and northern New England, western Washington, and in a few scattered areas.

A CORRECTION

In the last line on page 119 of the April 15 issue (number 4), "Division" should, of course, be Dominion, and the line should read "Dominion Laboratory of Plant Pathology."



Map I. Departure of Mean Temperature from the Normal for April 1946



Map II. Percentage of Normal Precipitation for April 1946.